REMARKS/ARGUMENTS

Claims 10-29 are active in the case.

Claims 1-9 have been canceled and new Claims 10-29 have been added. New Claims 10 and 11 are based on canceled Claims 7, 8 and 9 with the addition of the phrase "pharmaceutically acceptable salt, solvate or hydrate thereof", basis for which is found on page 5 of the specification. Changes in Claims 10 and 11 have been also made to provide proper Markush language and make the claims more readable. New Claims 12-29 have been added to preferred embodiments. Basis for new Claims 12-23 may be found on pages 4 and 5 of the specification. Basis for new Claims 24-29 may be found on pages 10 and 11 of the specification. No new matter has been added into the new claims.

It is requested that the Examiner acknowledge the claim to foreign priority made on August 8, 2001. Certified copies of the corresponding convention application(s) were submitted to the International Bureau in PCT Application No. PCT/JP00/00742. Receipt of the certified copy(s) by the International Bureau in a timely manner under PCT Rule 17.1(a) has been acknowledged in PCT/IB/304.

It is further requested that the Examiner consider the Form PTO-1449, filed September 2, 2003. It is requested that the Examiner consider the reference cited therein, initial the Form PTO-1449 in the appropriate box, sign and date the Form PTO-1449 and supply a copy to Applicants with the next Official Action.

The rejection of Claim 4 under 35 U.S.C. §101 is traversed.

The cancellation of Claim 4 now renders this rejection moot.

The rejection of Claims 1-3, 5 and 6 under 35 U.S.C. §102(a) as anticipated by Girlanda-Junges et al (u) or (v) is traversed. With the cancellation of Claims 1-9 and the

limitation of the claims to the subject matter of non-rejected Claims 8 and 9, this rejection is now moot.

The rejection of Claims 1-3 and 5-9 under 35 U.S.C. §103 as unpatentable over Borg and Girlanda-Junges et al is traversed.

Neither Borg nor Girlanda-Junges et al teach or suggest the use of the compound of the present claims in a method for the treatment of amyotrophic lateral sclerosis or a disorder caused by mutation in a superoxide dismutase gene. Girlanda-Junges et al only postulate that cyclohexenonic long-chain fatty alcohols may potentially lead to a viable therapy to alter the pathogenesis of neurodegenerative diseases. This postulation does not teach or suggest the use of the compound of the present claims in a method of treating amyotrophic lateral sclerosis or in the treatment of a disorder caused by a mutation in superoxide dismutase gene, as in the present claims.

Further, Borg in column 1, lines 45-63 merely identify amyotrophic lateral sclerosis as a disease arising from the consequence of the progressive disappearance of certain neurons and does not teach or suggest a method for the treatment of amyotrophic lateral sclerosis or a disorder caused by mutation in a superoxide dismutase gene, as in the present claims. The citation of column 13(a)(c) in Borg referred to by the Examiner merely raises the possibility of treatment of neuronal degeneration, in particular, during the course of neurodegenerative diseases(a) and to the possible treatment of various diseases, specifically excluded from which is amyotrophic lateral sclerosis or a disorder caused by mutation in a superoxide dismutase gene, as in the present claims.

Amyotrophic lateral sclerosis is a disease that is pathologically known for its extraordinarily unique behavior, as explained in detail on pages 1 and 2 of the present specification. Because of such predictable behavior, there is no teaching or suggestion in

either Girlanda-Junges et al or Borg that amyotrophic lateral sclerosis can be effectively cured by treatment with the compound of the present claims. The present inventors were the first to discover that the compound of the present invention is surprisingly effective against amyotrophic lateral sclerosis, based on the results of survival tests made on transgenic mice expressing a mutation in a superoxide dismutase gene demonstrated in Test Examples 1-3 of the specification. These mice are well known as an animal model for amyotrophic lateral sclerosis. Therefore, based on the above arguments, the claims distinguish over the combination of references.

Further, newly cited U.S. 6,228,893 discloses that a compound similar to or identical to the presently claimed compound, possesses neurite growth stimulating effects. However, the reference does not teach or suggest the treatment of amyotrophic lateral sclerosis by the use of a compound of the present claims, which is a primary objective solved by the present invention. As discussed above, amyotrophic lateral sclerosis is a disease that is known for its extraordinarily unique pathological behavior, and, because of such behavior, it is unpredictable whether this disease could be effectively cured by the method of U.S. 6,228,893, since the reference method merely discloses neurite growth stimulating effects in vivo. Since the present inventors were the first to discover that the compound of the present claims is surprisingly effective as a treatment against amyotrophic lateral sclerosis, based on the results of the survival tests in the specification made on transgenic mice expressing a mutation in the superoxide dismutase gene, and the fact that these mice are well known as an animal model for amyotrophic lateral sclerosis, it is submitted that the claims distinguish over U.S. 6,228,893, also.

It is submitted that Claims 10-29 are allowable and such action is respectfully requested.

Respectfully submitted,

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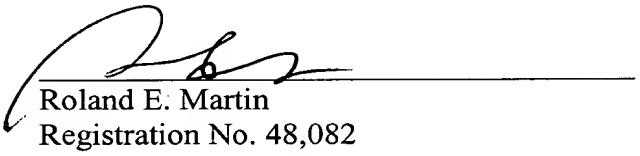

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